

Prevention of Accidental Breathing of Anoxic Gas Mixtures During Anesthesia

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ANOXIC anoxia is always a hazard to anesthetized patients when an anesthesia gas machine is used. One cause of inadvertent anoxia, the connection of an erroneously placed cylinder of gas to the oxygen yoke has been eliminated (barring manufacturers' error*) by the introduction of the pin-indexing system. The hazard of failure of the oxygen supply from other causes has not been eliminated. These include: (1) Exhaustion of the oxygen cylinder. (2) Insufficient opening of the oxygen cylinder to permit a free flow of gas after the pressure falls. (3) Failure of gas pressure in a piped oxygen system. (4) Failure to open the valve of a piped oxygen system. (This may not be recognized because of the flow of gas from a cylinder source which is subsequently closed.) (5) Nonpositive lock between oxygen piping and gas machine. (6) Failures in reducing valve or gas manifold. (7) Inadvertent disturbance of the setting of the oxygen flow meter.

Instances of most of the above causes of oxygen failure have been observed over the past eleven years. Presumably they occur in all hospitals. We have seen 100 per cent ethylene administered when an oxygen cylinder was exhausted. In another patient, a partially opened cylinder failed to deliver gas

*Holmdahl, M. H.: personal communication, 1961.

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when the pressure within it had fallen to 1,300 pounds per square inch. The result was the delivery of 100 per cent nitrous oxide to a semiclosed circle system. Each of these instances resulted in the death of the patient. In a third instance, the oxygen pipe line valve was inadvertently turned to the "off" position. The heart of this patient stopped beating due to anoxia. Fortunately, the error was recognized in time, resuscitation was successful and the patient recovered. Other "near misses" not resulting in cardiac arrest, but productive none the less of anoxic episodes, have occurred.

Case Reports

CASE 1: A middle aged woman was admitted for cholecystectomy. Her past history and physical examination were non-contributory.

Premedication was morphine sulfate 10 mg. and scopolamine 0.5 mg. Anesthesia was induced with 600 mg. of thiopental in intermittent doses. The patient was given ethylene and oxygen to breathe, and curare (15 mg.) in two divided doses was injected intravenously approximately twenty minutes after the induction. Tracheal intubation was attempted, but was unsuccessful. The mask was re-applied, but at this point the oxygen tank had become exhausted and the patient received 100 per cent ethylene. Severe hypotension resulted and the empty tank was discovered. Attempts to connect to a wall oxygen source were unsuccessful at first and

the patient was apneic for several minutes. Cardiac arrest occurred, the chest was opened, and cardiac massage undertaken. Oxygen was finally administered by positive pressure through an intratracheal tube after a delay of some minutes. Resuscitation of the heart was accomplished within two minutes of thoracotomy, but the period of anoxia was probably at least five or six minutes. Although there was no further difficulty with the circulation, the patient did not react at the termination of the procedure. She became febrile and convulsed. She died approximately 46 hours postoperatively.

CASE 2: An obese middle aged negro woman with a breast mass had a history of hypertension and had had several previous minor operations. An electrocardiogram was normal.

Premedication was secobarbital 100 mg., meperidine 75 mg. and scopolamine 0.5 mg. The patient was described as dry, calm, and sleepy with a blood pressure of 128/80. Anesthesia was induced with thiopental in fractional doses totalling 275 mg. Nitrous oxide and oxygen were administered by semi-closed circle absorption system. Approximately twenty minutes after induction of anesthesia just before the incision was made she became apneic. At this point it was noticed that the oxygen cylinder gauge, which shortly before had registered 1,300 pounds per square inch, had fallen to zero. Oxygen was not flowing and the patient had breathed 100 per cent nitrous oxide for an unknown period of time. The cylinder was reopened and was, in fact, found to contain 1,300 pounds per square inch pressure. Inflation of the lungs with oxygen resulted in an immediate return of respiration and a tachycardia of 160 with a blood pressure of 200/100 recorded immediately after reinflation. At no time was there evidence of cardiac arrest. Because of the rapid return of the vital signs to normal, as well as return of the widely dilated pupils to normal diameter, operation was undertaken and a benign breast cyst was removed.

Postoperatively, however, the patient did not recover consciousness and developed decerebrate rigidity. She expired eight and one-half months later without recovering consciousness.

Investigation of the size D cylinder involved in the incident disclosed that partial opening of the cylinder valve permitted the delivery of gas through a standard oxygen pressure regulator until the cylinder pressure dropped. The delivery pressure then fell to zero. This fault could be remedied by opening the cylinder further. It was established subsequently that this phenomenon occurred with most oxygen cylinders provided that they were opened to that critical point at which delivery of gas at high pressure just began.

CASE 3: The patient was a young Negro boy admitted for excision of a cyst of the knee. Past history and physical examination were unremarkable.

The patient was premedicated with secobarbital 40 mg., meperidine 38 mg. and scopolamine 0.4 mg. Following induction of anesthesia with open cyclopropane, anesthesia was continued with nitrous oxide and oxygen and tracheal intubation accomplished. The patient was then moved from the induction room to the operating room, and the gas machine connected to the wall oxygen. The cylinder oxygen was turned off. Approximately 30 minutes following induction, a pulse could not be felt. The wall oxygen had ceased to function and the patient was receiving 100 per cent nitrous oxide. The chest was opened immediately and the oxygen supply restored. A regular systolic contraction of the heart was restored within two minutes following the discovery of cardiac arrest. Respiration was controlled throughout this period of time, but the circulation seemed to return to normal. It was necessary to reanesthetize the child with nitrous oxide and oxygen approximately 45 minutes subsequent to the thoracotomy to permit closure of the chest.

In the immediate postoperative period, the patient remained comatose with marked flexor spasticity, shivering, and occasional tonic seizures. The reflexes were hyperactive and bilateral Babinski signs were present early. Although the patient spoke a few words on the first postoperative day, his state of consciousness fluctuated through the next two days and he was comatose most of the time. Following this interval, he made progress each day and left the hospital 19 days after the arrest.

His preoperative mental status never had been adequately evaluated. Psychological testing prior to discharge from the hospital revealed defects in response of organic pattern. Subsequently, the mother has commented that his mentality seemed unchanged, but that he had become a behavior problem. Psychological testing 14 months postoperatively revealed an I.Q. of 65, and the child was placed in a class for retarded children.

Discussion

The prevention of such incidents must be the responsibility of the anesthetist who should be alert to the setting of his flow meters and the biological responses of the patient. That this human control may be inadequate under the pressures of multiple responsibilities during the conduct of anesthesia is demonstrated by the incidents reported. It seemed desirable to secure mechanical assistance to aid the anesthesiologist. One method is the inclusion in gas machines of oxygen alarms which signal reduction of the oxygen flow. We are convinced that although these may be useful, they are necessarily subject to frequent false alarms and that in time the user may become conditioned to ignore the signal. Also these signals require an appropriate operator response. We believe that a suitable system for the prevention of anoxic accidents must be foolproof, independent of the active intervention of the anesthetist, and must operate in a "fail-safe" manner.

A device which meets these criteria has been developed by Arnold S. J. Lee. It works as follows (fig. 1):

Oxygen and all other gases to be combined into a predetermined breathing gas mixture are simultaneously controlled as to their individual flow rates by a single (MASTER) control pressure. Since the percentage of oxygen in the mixture is of paramount importance, oxygen pressure is used as the control pressure. This pressure, varied easily by a single knob, is employed in place of the customary spring in the delivery (SLAVE) regulators for all gases including oxygen. In this way, should the oxygen pressure change, the flow rates of all component gases automatically change proportionally. Therefore, the compo-

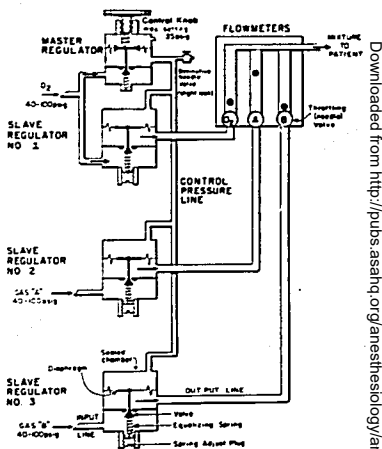


FIG. 1. "Master-slave" proportioning regulator system.

sition of the breathing gas mixture remains substantially constant. Should the oxygen input pressure fall to zero, the diaphragms of all slave regulators close and gas flow ceases.

The instrument also provides single knob control of the total flow-rate of gas mixture to nonbreathing systems. At some intermediate setting of the control knob the flowmeter needle valves are adjusted so that the flowmeters indicate the desired flow rates of the component gases. As the control knob is adjusted, the control pressure and hence the output pressures of the slave regulators vary in consonance. Since the flow rate through a needle valve is approximately proportional to the upstream pressure, the flow rates of all the component gases vary proportionally to the oxygen. For highest precision over a wide range of flow it is necessary that the component gases be supplied to the apparatus at a pressure substantially above atmospheric, e.g. seven atmospheres. The higher the pressure, the more accurate the control. At the customary 50 p.s.i. piped oxygen wall pressure, control accuracy adequate for anesthesia purposes may be achieved.

This system with rather simple equipment fulfills its function of safety in all anesthetic systems. It has been in use in our hospital for the last year and a half and has proved completely satisfactory. Although we are not aware of any incident in which the fail-safe feature has been called into play during clinical anesthesia because of oxygen failure, the prompt and efficient functioning of the device has been demonstrated deliberately to residents and visitors on hundreds of occasions without a single malfunction. The single knob control of total flow rate has proved particularly useful in adjusting the rate of inflow to pediatric nonbreathing systems without a need to adjust two or more flow meter settings for each desired change in total flow.

Summary

Three cases of death or cardiac arrest due to the inadvertent breathing of anoxic mixtures have been reported with a discussion of the sources of such error and the desirability of its prevention. Death or serious injury due to technical errors indicate the urgent need to make anesthesia apparatus foolproof. Complete prevention of the delivery of oxygen deficient mixtures due to loss of pressure is possible by a simple modification of conventional gas machines, and a "master-slave" proportioning regulator system is described. The device is in active use and produces no complication of the day-to-day administration of anesthesia.

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MONITORS The electrocardiograph reports only the electrical events taking place in the heart and cannot represent functional competence. It is not designed to supply the valuable information that can be secured from the pulse, regular recording of the blood pressure, and an assessment of color, perspiration, venous pressure, etc. Other devices dependent on the R wave of the electrocardiograph complex for actuation of visual or auditory signals can reflect only one activity of the heart, its rate. These units have been known to signal even with a heart in standstill. An esophageal stethoscope provides an estimate of cardiac function. Photoelectric cells, which are activated by changes in density due to blood flow in the finger, can measure functional activity, but these are susceptible to artifacts of movement. The electroencephalogram shows changes which can often be more easily detected by the observation of traditional signs such as pupillary size and reaction to light. Dynamic carbon dioxide analyzers and instruments for measurement of the pH, P_{CO_2} , and P_{O_2} of blood cannot be considered ordinary monitors. Instruments such as the ventimeter and airway pressure gauges are to be recommended for ordinary clinical use. (Cullen, S. C.: *Monitors and Clinical Judgment*, *Amer. J. Surg.* 102:300 (Aug.) 1961.)